Infra-His Bundle Origin of Bidirectional Tachycardia

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SUMMARY

A case of bidirectional tachycardia is presented in a patient with cardiomyopathy, pulmonary emboli, and digitalis toxicity. The arrhythmia has usually been considered ventricular in origin. A supraventricular origin has also been suggested. Simultaneous recordings of standard electrocardiographic leads and His bundle potentials demonstrated that the abnormal rhythm originated in the left ventricle and indeed was a bidirectional ventricular tachycardia. Enhanced phase 4 depolarization in the divisions of the left bundle branch is advanced as a theoretic underlying mechanism in some instances of this arrhythmia.

Additional Indexing Words:

Aberrant ventricular conduction Phase 4 depolarization Digitalis toxicity Hemiblock

BIDIRECTIONAL tachycardia is an infrequently observed arrhythmia characterized by alternating rightward and leftward axis shifts. There is a regular, or slightly irregular, ventricular rate of 140–180 beats/min. The associated electrocardiographic complexes are stated to have a constant right bundle-branch block pattern.

Seventy-two cases have been recorded in man since the first description by Schwensen in 1922.[1–31] However, the mechanism of this arrhythmia remains controversial.[30] We have obtained intracavitary electrograms in a patient with bidirectional tachycardia which revealed an infra-His origin of this arrhythmia. We propose that enhanced phase 4 depolarization is a plausible underlying mechanism in some instances of bidirectional tachycardia.

Case Report

The patient, a 59-year-old male clerk, was admitted to the hospital for evaluation of dizziness and shortness of breath. Seventeen and five years prior to admission the patient had episodes of "fluttering" heart beat. On the latter occasion, an electrocardiogram revealed atrial fibrillation with a ventricular response of 150 beats/min. The arrhythmia was slowed with digitoxin and then converted to sinus rhythm. Digitoxin was continued at a dose of 0.15 mg daily. Subsequently, annual employment examinations revealed an irregular pulse.

Two months prior to admission, the patient had a "flulike" syndrome characterized by weakness, anorexia, nonproductive cough, vomiting, and diarrhea. One month prior to admission, atrial fibrillation was noted at a ventricular response of about 130 beats/min. The dose of digitoxin was increased to 0.30 mg daily for 2 weeks and then was reduced to daily doses of 0.30 mg alternating with 0.15 mg. Five days prior to admission, persistent symptoms of dizziness and shortness of breath occurred.

Physical examination on admission revealed mild respiratory distress with a respiratory rate of 20 breaths/min. The blood pressure was 90/70 mm Hg, the temperature was normal, and there was an irregular pulse of 150 beats/min. The examination of the neck, with the trunk elevated at an angle of 45°, revealed jugular venous distension. The thyroid was of normal size. The examination of the chest revealed moist rales at both lung bases. The cardiac impulse was felt at the fifth intercostal space in the anterior axillary line. The heart sounds were faint and there were no murmurs. The examination of the abdomen was normal. The femoral and popliteal pulses were normal. There was no leg edema or phlebitis.

Laboratory data revealed a normal complete blood count. Creatinine, sodium, potassium, chloride, total bicarbinate, arterial blood gases, and *p*H were also normal. The serum digitoxin level was 54 ng/ml (therapeutic range 5–30 ng/ml). Chest X-ray revealed upper zone vascular distension and generalized cardiomegally.

The hospital course was complicated by persistent heart failure and a variety of transient, abnormal heart rhythms. The most prevalent rhythm, however, was atrial fibrillation with a rapid ventricular response which ranged from 95 to 150 beats/min.

During the first 10 days the serum digitoxin level gradually fell from 54 ng/ml to 30 ng/ml. On the fifteenth hospital day, right flank and pleuritic chest pain was experienced. A pulmonary arteriogram was

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Supported in part by a Training Grant from the National Institutes of Health, HL-5909.

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Received September 11, 1972; revision accepted for publication February 12, 1973.

diagnostic of bilateral pulmonary emboli. Anticoagulation with heparin was then instituted and sodium warfarin was substituted for heparin during the latter part of the hospitalization.

The remaining hospital course was complicated by rapid atrial fibrillation and left ventricular decompensation. Cardiac catheterization, performed on the twenty-eighth hospital day, revealed right and left heart failure. The cardiac index was 0.9 liters/min/m^2. Left ventricular angiography demonstrated poor contractility of the left ventricle and minimal mitral regurgitation.

The patient died on the thirty-second hospital day. Post mortem examination of the heart and lungs revealed a cardiomyopathy with right and left ventricular hypertrophy and dilatation. The coronary arteries and cardiac valves were normal. Examination of the lungs revealed recent and old bilateral pulmonary emboli.

Electrocardiograms and His Bundle Electrograms

The admission electrocardiogram (fig. 1) revealed a slightly irregular ventricular rate of 150 beats/min. There were sequences of right bundle-branch block pattern beats with an electric axis that alternated between −90° and +120°. Atrial fibrillation was apparent after the ventricular rate was diminished to 130/min by intravenous lidocaine (100mg). Each of the alternating abnormal patterns also manifested as a single abnormal complex. The cycle terminating in an abnormal complex was preceeded at times by a long cycle length and at other times by a short cycle length.

A His bundle study was performed by the usual technic[32] in order to better characterize the arrhythmia. As the catheter was withdrawn from the right ventricular cavity, a right bundle-branch potential was identified. It was separated from normal ventricular activation by a short time interval. A well defined triphasic His bundle potential[32–36] was then identified and characterized as 20 msec in duration and 53 msec before each normally conducted beat. A compelling reason not to attempt validation by bundle of His pacing[37] was the likelihood of producing repetitive ventricular responses while introducing high-output stimuli within the right ventricle in the setting of digitalis toxicity.[38–40] All normal complexes were preceded by a His bundle potential. Abnormal complexes, however, were not preceded by a His bundle potential (figs. 2, 3). Stimultaneous standard leads II and V_1 demonstrated that the abnormal complexes were all of right bundle-branch block configuration, and that there was an alternating electric axis. There was a variable pattern of right bundle-branch block in lead V_1. On each occasion the complex that initiated a sequence of abnormal beats had a right

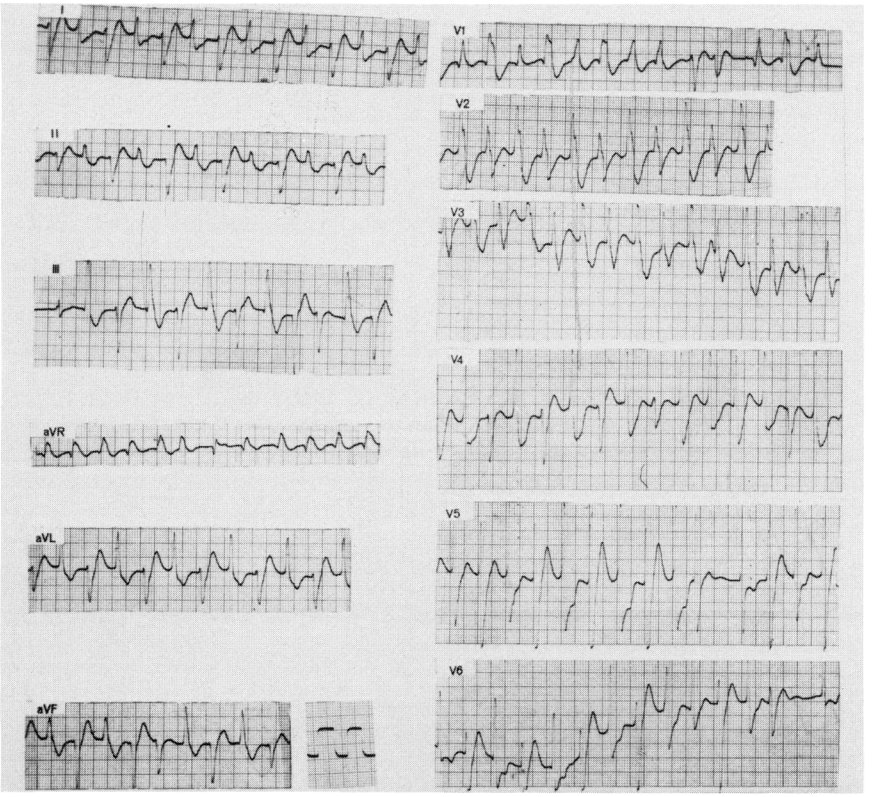

Figure 1

Admission ECG. Bidirectional tachycardia is present. For the most part the beats alternate between an axis of −90° and +120°. There are occasional beats of normal appearance such as the first beat in lead III and the nonright bundle-branch block beats in lead V_1.

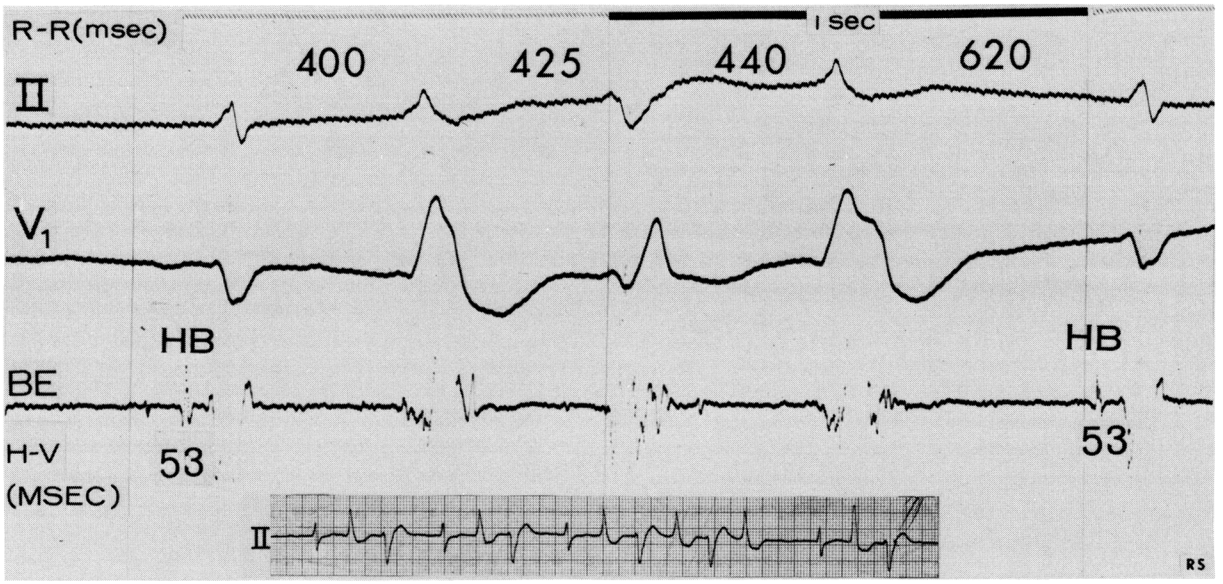

Figure 2

His bundle electrogram during bidirectional ventricular tachycardia. Recordings are of simultaneous standard leads II and V_1, and a bipolar electrogram from the area of the bundle of His. The first and last beats are normally conducted and are preceded by a His bundle potential (HB) *with a H-V time of 53 msec. There is an intervening sequence of three abnormal beats which are not preceded by His potentials. The abnormal beats alternate their axis and have a right bundle-branch block pattern. The intervals between abnormal beats are slightly variable. There is a bottom insert which illustrates the intermittency of the tachycardia at the time of His bundle study. H-V = His-ventricular, an indicator of intraventricular conduction time. (Normal = 35–50 msec.)*

bundle-branch block pattern with right-axis deviation. Two examples among many are shown in figures 2 and 3.

Clinical Features of Bidirectional Ventricular Tachycardia

A summary of case material since 1922 is presented in table 1. The arrhythmia occurred in a clinical setting of severely compromising cardiac disease. Atrial fibrillation was present in 44% of patients (32/72). Digitalis was being administered in 82% of patients (59/72) and, in many, was stated to be a causative agent in producing bidirectional tachycardia.[1–6, 10, 11, 13, 15, 18, 23, 26] In thirty-nine patients whose clinical course was reported,[29, 30] 70% (27/39) expired within hours to days, 15% (6/39) expired within 3–12 months, and only 15% (6/39) survived more than 1 year. Autopsy analysis revealed varied cardiac pathology such as pericarditis,[3, 6, 7, 12, 19] myocarditis,[28, 31] rheumatic heart

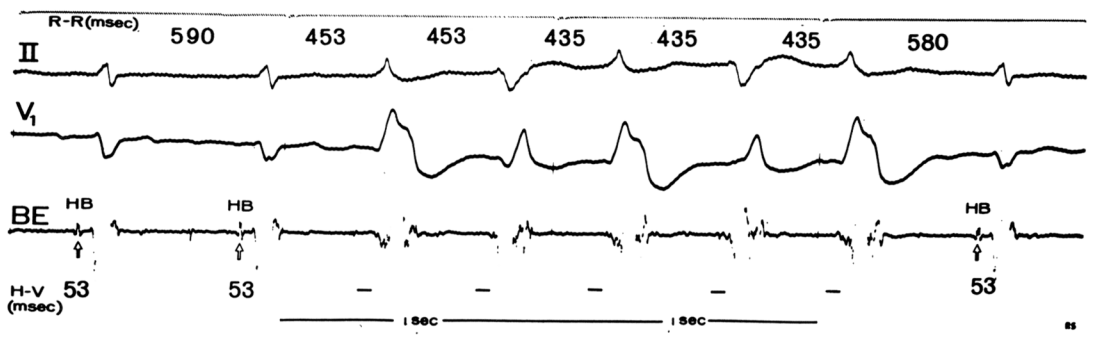

Figure 3

His bundle electrogram during bidirectional ventricular tachycardia. The first, second, and last beats are normally conducted and are preceded by His bundle excitation (HB) *with an H-V time of 53 msec. There is an intervening sequence of five abnormal beats which are not preceded by His bundle potentials. The abnormal beats alternate their axis and all have a right bundle-branch block pattern. The intervals between the first and second abnormal beat is longer (453 msec) than those which follow (435 msec). V-V = His-ventricular, an indicator of intraventricular conduction time. (Normal = 35–50 msec.)*

Table 1

Some Clinical and Electrocardiographic Features of Bidirectional Ventricular Tachycardia

Ref	Rhythm preceeding or following BVT: AF	NSR	Other	Digitalis	Axis of initiating complex in BVT
1	+			+	LAD
2	+			+	
3	+			+	
3	+			+	LAD, RAD
3			+	+	
4	+				
4	+				
4		+		+	
4		+			
5		+		+	
6	+			+	RAD
6		+		+	RAD
6		+		+	LAD, RAD
6	+			+	RAD
6		+		+	
7			+	+	
8			+	+	
8	+			+	RAD
9			+	+	
10	+			+	
11	+			+	
12			+	+	
12	+			+	
13	+			+	LAD, RAD
14	+			+	LAD
15	+			+	
16	+			+	LAD
17	+			+	
18	+			+	
19	+			+	LAD
20	+				
20		+			
20		+			
21	+			+	
22	+			+	LAD
23	+				
24	+			+	
25	+			+	
25			+	+	
25			+	+	
25			+	+	
26*	4/10	2/10	4/10	8/10	RAD (2)
27	+			+	LAD
28		+		+	
28		+			LAD
28		+		+	
29		+		+	LAD
29			+	+	
30	2/13		11/13	12/13	LAD (1)
31		+			LAD
31		+			
Totals 72 patients	32	16	24†	59	LAD = 13 RAD = 9

Circulation, Volume XLVII, June 1973

disease,[12, 19] myocardial infarction,[17] and vacuolar degeneration of myocardial cells.[6, 7, 12, 28, 31]

Discussion

This patient is similar to the majority of patients with bidirectional tachycardia. There was documentation of severely compromised cardiac function. The initial impression of digitalis toxicity was confirmed by a substantially elevated digitoxin level. Death occurred during the hospitalization.

Bidirectional tachycardia has been a well documented abnormality for 50 years. The proposed mechanisms for this arrhythmia have been recently reviewed.[30] The two most favored theories include a supraventricular origin with aberrant ventricular conduction and ectopic impulse formation within the left ventricle.

Indirect evidence that bidirectional tachycardia originates from a supraventricular focus comes from the association of this arrhythmia with A-V nodal or junctional tachycardia.[16, 19, 25, 29] Also, bidirectional tachycardia has been abolished with vagal maneuvers.[16, 19, 22] Rosenbaum believes that bidirectional tachycardia is a supraventricular tachycardia with permanent aberrant conduction in the right bundle branch.[30] The alternating axis shifts result from alternate aberrant conduction in the two divisions of the left bundle branch.[30]

The repetitive right bundle-branch block pattern that occurs with aberrant conduction from a supraventricular pacemaker site is believed to be the result of a perpetuation of refractoriness in the right bundle branch.[41, 42] Interference with normal excitation of the anterior division of the left bundle branch results in the late depolarization of the anterolateral wall and a leftward shift of the electrical axis.[43–50] Interference with normal excitation of the posterior division of the left bundle branch results in a rightward shift of electrical axis.[44, 48, 49] Therefore, alternating blockade in the divisions of the left bundle branch will result in alternating rightward and leftward axis shifts of sequential aberrantly conducted beats. Alternating conduction of the left bundle branch divisions is believed possible because the blocked division is "insulated" from the delayed excitation which occurs in the area of its distribution thereby

*Cases were excluded that were previously reported in ref 25.

†Includes one patient who also had NSR (ref 7).

Abbreviations: AF = atrial fibrillation; Ref = reference; NSR = normal sinus rhythm; BVT = bidirectional ventricular tachycardia.

permitting total recovery in time to conduct the next impulse.[30] According to this view, the division which was previously conductive becomes refractory to the next impulse.[30]

His bundle electrocardiography provides more precise data than have heretofore been available for analysis of bidirectional ventricular tachycardia. Our data support an infra-His bundle origin of this puzzling arrhythmia. First, the His bundle recording demonstrated the absence of sequential His bundle-ventricular excitation during abnormal ventricular conduction. Second, there was evidence of two types of isolated ventricular ectopic activity which, when in sequence, resulted in a bidirectional arrhythmia. Third, the cycle terminating in an abnormal beat did not always follow a long cycle length, which is the cycling pattern usually observed in aberrant supraventricular conduction.[51] Fourth, the beat that initiated an abnormal sequence had a configuration of right bundle-branch block with right-axis deviation contrary to the expected right bundle-branch block left-axis deviation configuration commonly associated with aberrant supraventricular conduction.[30, 52]

In our view, a theoretic explanation for bidirectional tachycardia can be made by assuming that

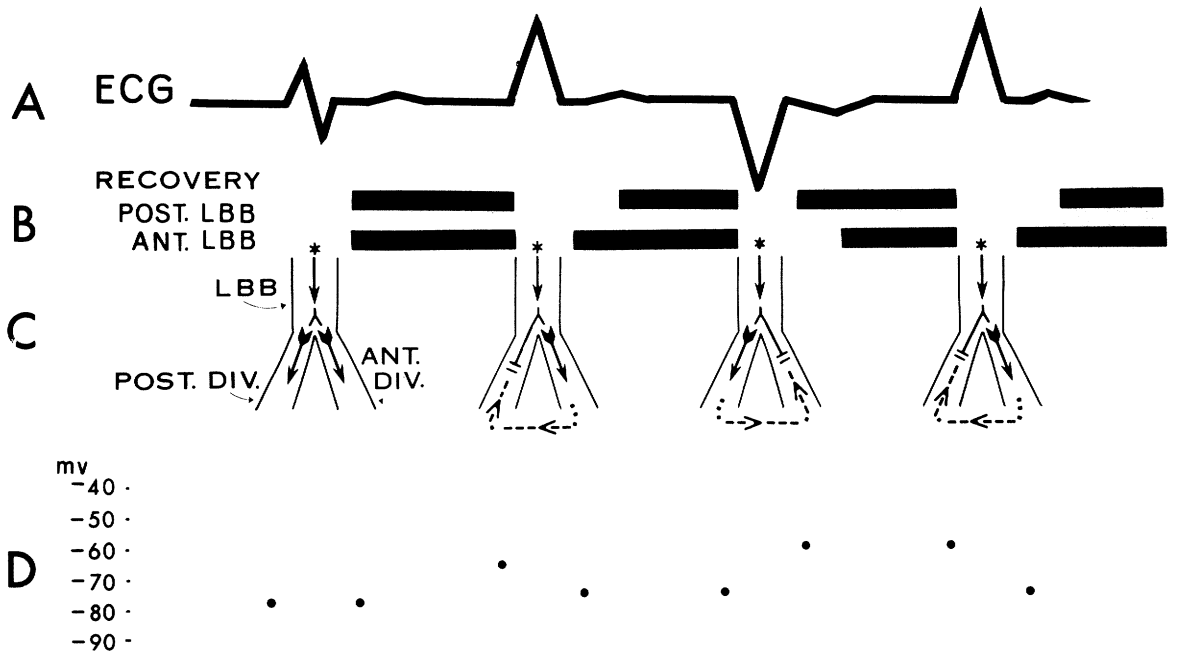

Figure 4

The mechanism of alternating axis deviation during bidirectional ventricular tachycardia: a schematic representation. (See text for additional explanation.) **(A)** *Electrocardiogram. The first beat is of normal configuration and is followed by three abnormal beats with respective right, left, and right-axis shifts. A right-axis shift results from blocked transmission in the posterior division of the left bundle branch. A left-axis shift results from blocked transmission in the anterior division of the left bundle branch.* **(B)** *Depicts the recovery time of each division. The earlier excited division has a longer recovery time than the blocked, tardily excited division. Thus, in the event of posterior division block in the second and fourth beats, the anterior division will have a longer recovery time than the posterior division following excitation.* **(C)** *Antegrade conduction from an ectopic pacemaker (*) in the common left bundle branch advances down the left bundle branch* (LBB) *and into its divisions. Conduction is normal in the first beat, conduction blocks in the posterior division in the second and fourth beats, and blocks in the anterior division in the third beat. In each instance of blocked division in a retrograde fashion (interrupted arrows).* **(D)** *Represents the transmembrane action potential (mv) of the anterior and posterior division at the time of antegrade excitation. The transmembrane action potential is represented by a dot under the appropriate division. After the first normal beat, the transmembrane action potential rose to —65 mv in the posterior division because of enhanced phase 4 depolarization and resulted in transmission block. The anterior division was conductive and had a longer diastolic recovery period than the tardily excited blocked posterior division. The longer diastolic recovery period permitted a longer interval for phase 4 depolarization in the anterior division which reached —60 mv and produced block at the time of antegrade excitation. A concommitant leftward axis shift occurred. The last beat again has block of the posterior division because of a perpetuation of differential recovery times and phase 4 depolarization.*

there is an ectopic ventricular pacemaker, enhanced phase 4 depolarization (the spontaneous loss of diastolic resting membrane potential) in the divisions of the left bundle branch and retrograde invasion of the blocked division of the left bundle branch. A schematic representation of these variables is presented in figure 4. Digitalis, hypoxia, and cardiac dilitation are frequently associated with this arrhythmia and are known to enhance phase 4 depolarization.[53–56] Singer has shown that phase 4-related block may be localized to only one area of a conductive pathway.[55] The failure of propagation of a wave front of excitation can occur when excitation advances to conducting tissue with abnormal resting membrane potentials in the range of −65 to −60 mv.[55] If we assume a steeply increased rate of rise of phase 4 depolarization, when threshold potential is reached, ectopic activity will occur. If one left bundle division has approached −65 to −60 mv more rapidly than the other at the time of an advancing excitation wave from an ectopic focus, there will be blockade of the wave front in that division. Moreover, the other division could be excitated with a variable transmission time depending upon its transmembrane potential. The reader is referred to the legend of figure 4 for a detailed explanation of alternating axis.

Alternating antegrade block in the divisions of the left bundle branch, because of enhanced phase 4 depolarization, could explain bidirectional ventricular tachycardia in those cases where the pacemaker is supraventricular, at the A-V node, in the bundle of His, in the common left bundle branch as presented in figure 4 for illustrative purposes, or at some appropriate location in the left ventricle such as the junction between the anterior and posterior divisions of the left bundle branch.

The abnormal right-axis complexes had a taller and broader R wave in lead V_1 than did the abnormal left-axis complexes. The H-V time was constant at 53 msec and was only noted with normal beats. Fusion beats were not noted. Variations in the pathways of excitation from an ectopic pacemaker[57, 58] could account for the nonuniformity of right ventricular excitation and could explain the two general variations in the right bundle-branch complexes in lead V_1.

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